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Open heart surgery in first year of life using profound hypothermia (core cooling) and circulatory arrest[1]
Experience with 134 consecutive cases

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SUMMARY Between April 1970 and December 1977, 134 infants aged 2 days to 12 months underwent open heart surgery using profound hypothermia and total circulatory arrest. The technique of bypass (core) cooling is described. Results are reviewed for 4 principal diagnoses: 'simple' transposition of the great vessels, total anomalous pulmonary venous connection, ventricular septal defect, and Fallot's tetralogy. A mortality of 44 per cent in 32 cases during the first 3-year period has been reduced to 22 per cent in 102 cases during the subsequent 5 years. The overall mortality for the entire period was 28 per cent. The policy for the management of each diagnostic group is outlined.

The incidence of congenital heart disease is approximately 8 per 1000 live births (Rudolph, 1974). A third of this number of infants has critical congenital heart disease which leads to either cardiac investigation, surgery, or death (Nadas *et al.*, 1973). During the past decade, rapid and exciting progress has been made and early corrective surgery for the majority of patients has been advocated by many surgeons (Barratt-Boyes, 1973; Cartmill *et al.*, 1973; Venugopal *et al.*, 1973; Bailey *et al.*, 1976). This achievement results from improvements in the application of the techniques of profound hypothermia and cardiopulmonary bypass and advances in pre- and postoperative management. A simple method of bypass (core) cooling followed by circulatory arrest and bypass rewarming has been employed at the Royal Liverpool Children's Hospital since 1970 for the correction of congenital heart defects during the first year of life (Hamilton *et al.*, 1973). In this report, we outline this technique and present the results of our experience.

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Method

Anaesthesia is induced with thiopentone and d-tubocurarine chloride and is maintained with nitrous oxide and oxygen (N_2O 4 l/min, O_2 2 l/min) to which carbon dioxide is added to give a final concentration of 2 per cent at temperatures above 35°C and 5 per cent below 35°C. Standard methods of monitoring are employed during and after operation. Supplementary doses of d-tubocurarine chloride and phenoxybenzamine (1 mg per kg intravenous) are given just before starting perfusion. The prime consists of 500 ml of fresh heparinised blood, 200 ml Hartmann's solution, 50 ml 5 per cent dextrose, and 3 ml 8·5 per cent sodium bicarbonate. Before perfusion, blood is maintained at a temperature of 37°C and is equilibrated with 95 per cent oxygen and 5 per cent carbon dioxide. The proportion of carbon dioxide is increased to 10 per cent at 30°C.

The Temptrol infant model Q130 oxygenator is used in conjunction with an infant heat exchanger in the arterial side of the bypass circuit. Perfusion is carried out at a rate of 2·4 l/min per m^2. A median sternotomy is used except for the correction of total anomalous pulmonary venous connection for which we favour a left anterolateral thoracotomy with trans-sternal extension. A short vertical pericardiotomy is made to expose only the upper part of the heart and the roots of the great vessels. This permits cannulation of the aorta and the right atrial appendage and maintains the normal position of the heart until perfusion is established. We believe that this reduces the likelihood of ventricular fibrillation which can result from kinking of the coronary arteries if the heart is allowed to tilt upwards when the pericardial sac is opened widely initially.

Once perfusion is established, the temperature of the perfusate is gradually reduced. A temperature differential of 10°C is maintained between the patient and the water circulating through the heat exchanger. A rate of cooling between 1 and 0·5°C per minute is achieved. Should the differential between nasopharynx and oesophagus exceed 5°C, the rate of cooling is reduced. During the cooling period, the pericardial sac is opened widely and the anatomy of the heart is studied. When the patient's temperature reaches the predetermined level, usually 18°C, perfusion is continued for a further 5 minutes at that temperature to reduce temperature gradients. This minimises the tendency for the temperature to drift upwards during the period of circulatory arrest. On average, the predetermined temperature is reached after 30 to 35 minutes of perfusion (core cooling).

At this stage, the arterial input is stopped, the line clamped, and the patient exsanguinated into the oxygenator via the right atrial cannula. The aorta and the pulmonary artery are occluded in a single clamp and the venae cavae are snared. During the period of circulatory arrest, blood in the oxygenator is maintained at the patient's body temperature. The intracardiac repair is completed within 45 to 60 minutes of circulatory arrest in the majority of cases. On the rare occasions when circulatory arrest is necessary for more than 70 minutes, a further short period of perfusion (5 to 10 minutes) is used before a second period of circulatory arrest. On completion of intracardiac surgery, rewarming perfusion is started by pressurising the arterial and venous systems. Air is removed from the aortic root by aspiration and from the left heart chambers via a left ventricular vent. When the patient's temperature reaches 30°C any base deficit or electrolyte imbalance is corrected. Rewarming is usually carried out at the same rate as cooling (0·5 to 1°C per minute). When the temperature reaches 34°C, perfusion is discontinued, provided that the cardiac output is adequate. Further rewarming is carried out by means of a water mattress beneath the patient. Usually, the rewarming period varies between 30 and 35 minutes.

Postoperatively, muscle relaxants are not reversed and the patients are ventilated through a nasotracheal tube connected to a square wave ventilator. The average period of ventilation is 30 hours.

Table 1 *Age distribution*

Age (mth)	*No. of patients*
0- 1	18
2- 3	30
4- 6	23
7-12	63
Total	134

Patients and results

During the period from April 1970 to December 1977, 134 infants in the first year of life underwent open heart surgery under profound hypothermia. There were 81 boys and 52 girls, ranging in age from 2 days to 12 months (mean 174 days); 71 were under 6 months of age (54%) (Table 1).

There were 4 principal diagnostic groups: simple transposition of the great vessels (31%), total anomalous pulmonary venous connection (19%), isolated ventricular septal defect (17%), and tetralogy of Fallot (9%). Less frequent diagnoses were pulmonary atresia with intact septum (7 patients) and aortopulmonary window (5 patients). Four patients had complex forms of transposition of the great vessels. There was also a miscellaneous group of 15 patients.

(1) 'SIMPLE' TRANSPOSITION OF GREAT VESSELS

Forty-two patients, 28 of whom were boys and 14 girls (mean age 6·3 months), had Mustard's

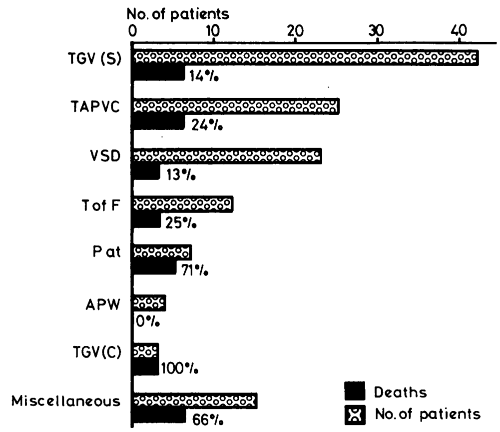

Fig. 1 *Hospital mortality related to type of cardiac defect (134 patients). TGV(S) = simple transposition of the great vessels; TAPVC = total anomalous pulmonary venous connection; VSD = ventricular septal defect; T of F = tetralogy of Fallot; P at = pulmonary atresia; APW = aortopulmonary window; TGV(C) = complex transposition of great vessels.*

Table 2 *Causes of hospital deaths after Mustard's operation for transpcsition of the great vessels*

Case no.	*Time of death*	*Cause*	*Postmortem findings*
1	1st day	Supraventricular arrhythmia	Satisfactory intracardiac repair
2	3rd day	Low cardiac output	Not available
3	10th day	Low cardiac output; renal failure; cerebral damage	Complete SVC obstruction due to organised thrombus
4	13th day	CHB low; cardiac output	IVC narrowed
5	5th day	Renal failure	Bilateral renal vein thrombosis
6	DOT	Sudden cardiac arrest	—

CHB, complete heart block; IVC, inferior vena cava; SVC, superior vena cava; DOT, death on the table.

operations using a pericardial baffle. There were 6 hospital deaths (14%) and 2 late deaths (Fig. 1). The causes of hospital death are shown in Table 2. The 2 late deaths were the result of pulmonary venous obstruction. One died 4 months later while awaiting a further operation and the other died 2 months later; necropsy showed pulmonary venous obstruction and bronchopneumonia.

Clinical follow-up for 3 months to 7 years after operation (mean 3·5 years) of 34 survivors (81%) shows that these children were progressing satisfactorily. Thirty-two patients were pink and fully active and 1 patient has minimal cyanosis at rest with exercise tolerance at the lower end of the normal range. Another patient required reinvestigation and reoperation 6 months later for pulmonary venous obstruction, and developed atrial flutter with 2:1 atrioventricular block and paralysis of the diaphragm. Four years later he was well despite some pulmonary venous obstruction. There was no clinical evidence of caval obstruction in the survivors.

Episodes of arrhythmia occurred in the immediate postoperative period in 16 of the 42 patients. Seven children remained in nodal rhythm with a satisfactory heart rate for periods varying from 24 hours to 7 days. One child had a sustained nodal tachycardia which reverted spontaneously to sinus rhythm after 36 hours. Junctional rhythm was present in 5 patients, 1 of whom died in the operating theatre without ever achieving an adequate cardiac output; 2 of the remaining 4 patients required temporary pacing. A further 2 patients had 2:1 heart block and also required pacing. One reverted to sinus rhythm after 7 days, but in the second the arrhythmia was probably a major contributing cause of death. All but 2 of the long-term survivors attending this hospital are in sinus rhythm. One child with nodal rhythm in the postoperative period now has first degree heart block (PR 0·24 s) and another who had complete heart block in the postoperative period now has a stable nodal rhythm.

(2) TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION

Twenty-five infants (16 boys and 9 girls) (age distribution is shown in Fig. 2) underwent total correction. Fifteen had supracardiac, 3 had cardiac, 2 had the mixed type, and 5 had infracardiac drainage. There were 6 hospital deaths (24%) and no late deaths (Fig. 1). The youngest, a 5-day-old infant, survived correction of the infracardiac variety of

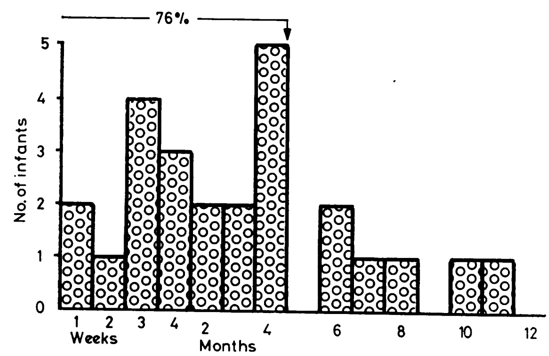

Fig. 2 *Age distribution of 25 infants with total anomalous pulmonary venous connection.*

Table 3 *Causes of hospital deaths after total correction of total anomalous pulmonary venous connection*

Case no.	*Type*	*Time of death*	*Cause*	*Postmortem findings*
1	Supracardiac	4th day	Extensive infarction of right lung	Good anastomosis
2	Infracardiac	DOT	Low cardiac output	Hypoplastic left heart chambers + aortic coarctation
3	Supracardiac	1st day	Low cardiac output	Hypoplastic left heart chambers
4	Mixed	DOT	Ventricular fibrillation (closing chest)	Not available
5	Cardiac	3rd day	Pulmonary oedema	Not available
6	Supracardiac	6th day	Severe cerebral damage	Diffuse brain softening

Table 4 *Causes of hospital deaths after patch closure of ventricular septal defect*

Case no.	*Associated anomalies*	*Time of death*	*Cause*
1	Multiple VSD and ASD (incomplete preoperative diagnosis)	48 hours	CHB; low cardiac output
2	ASD and PDA and LSVC to CS; peripheral branch PA stenosis (preoperative pneumonia not controlled)	4th day	Respiratory insufficiency
3	—	4th day	Low cardiac output

VSD, ventricular septal defect; ASD, atrial septal defect; PDA, persistent ductus arteriosus; LSVC, left superior vena cava; CS, coronary sinus; PA, pulmonary artery; CHB, complete heart block.

this lesion. The causes of death are summarised in Table 3. Mortality in infants with this lesion was not related to age, but rather to the anatomical type of venous connection. Follow-up from 5 months to 8 years (mean 3 years and 9 months) of 19 survivors (76%) shows that all of them are well and have normal exercise tolerance. One patient however, who had fits in the postoperative period, requires anticonvulsant therapy for convulsions with hyperpyrexia.

(3) VENTRICULAR SEPTAL DEFECT

There were 23 patients in this group, 12 boys and 11 girls. The mean age was 6·6 months. Three patients had associated atrial septal defect. Patch closure of the ventricular septal defect was affected via a right ventriculotomy in all cases. There were 3 hospital deaths (13%) and no late deaths. The causes of death are listed in Table 4.

(4) FALLOT'S TETRALOGY

Twelve patients, 6 boys and 6 girls, mean age of 7·0 months, had total correction, with 3 hospital deaths (25%) (Fig. 1). Gusset reconstruction was performed in 1 case. One patient died after failure of a pacemaker inserted for complete heart block at operation.

Another patient presented unusually in cardiac failure before operation and died after operation in a low cardiac output state. Necropsy showed myocarditis. A third patient died as a consequence of severe cerebral damage possibly as a result of intraoperative air embolism.

(5) OTHER LESIONS

Pulmonary atresia with intact interventricular septum

These infants underwent open heart surgery during the first week of life because of inadequate pulmonary blood flow. Five of these 7 patients died. When the pulmonary valve is thickened and the valve ring is of inadequate calibre, we have not met with success even when the valve tissue has been excised and a transannular gusset has been inserted. Under these circumstances and in the presence of hypoplasia of the right ventricle, we favour a combination

Table 5 *Miscellaneous group of infants who underwent totally corrective intracardiac surgery between April 1970 and December 1977*

Miscellaneous group	*No. of cases*	*No. of deaths*
Truncus arteriosus	1	0
Complete AV canal	2	2
Univentricular heart	1	1
Hypoplastic aortic arch and left ventricle	1	1
ASD (pulmonary hypertension)	2	0
Interrupted aortic arch (type B)	3	3
Tricuspid atresia	1	1
Cor triatriatum	1	1
Mitral regurgitation	1	0
Anomalous coronary artery	1	1
Aortic stenosis	1	0
Total	15	10

of Rashkind's septostomy and a systemic-to-pulmonary arterial shunt (Waterston's anastomosis).

Five patients with *aortopulmonary window* all survived, and 4 patients with *complex forms of transposition of the great vessels* all died.

In the miscellaneous group, 2 infants aged 8 and 11 months had *atrial septal defects* with pulmonary hypertension. Both were failing to thrive. One suffered from recurrent pulmonary infections and the other from congestive cardiac failure which was resistant to medical treatment. Both had pulmonary artery pressures in excess of 50 mmHg. These infants had large ostium secundum defects which required patch closure; both survived. A 4-month-old baby with *truncus arteriosus* (*type II*) had a successful correction. No other patients with more complex lesions survived in this group (Table 5).

Summary of mortality

Thirty-seven patients died within 30 days of operation (28% hospital mortality). There were 2 late deaths (late mortality 1·5%). The distribution in each year of the patients operated on and the related hospital mortality is shown in Fig. 3. The hospital mortality of 44 per cent in 32 cases during the first 3 years has been reduced to 22·5 per cent in 102 cases during the subsequent 5 years. The

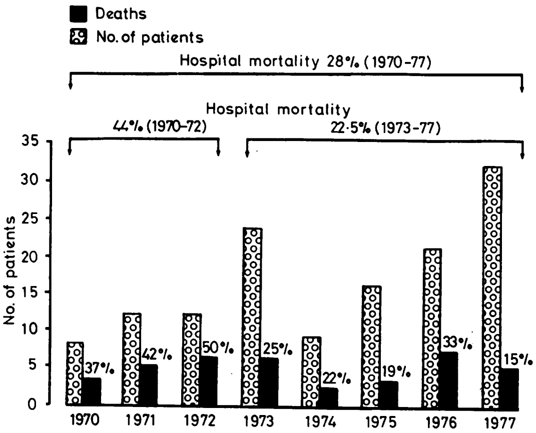

Fig. 3 *Annual distribution of patients and hospital mortality in 134 infants.*

hospital mortality was 50 per cent in the first month of life (mean age 10 days), 33 per cent in the second and third months of life (mean age 63 days), and 35 per cent at 4 to 6 months (mean age 141 days). The overall mortality rate for the group of patients operated on within the first 6 months was 38 per cent. The mortality over 6 months (mean age 9·8 months) was 17 per cent (Fig. 4).

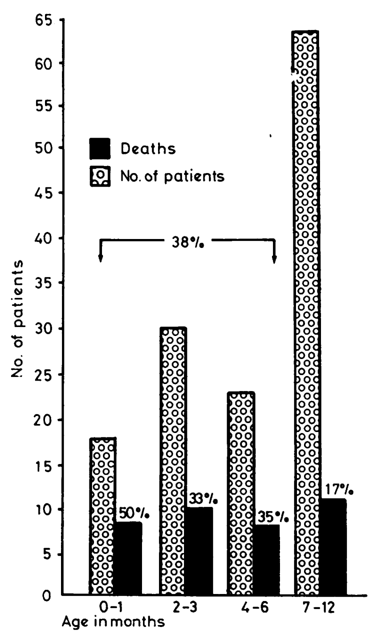

Fig. 4 *Age distribution and hospital mortality in 134 infants.*

Discussion

Although encouraging results using conventional cardiopulmonary bypass techniques for the correction of congenital heart disease in infancy have been reported (Gersony *et al.*, 1971; Breckenridge *et al.*, 1973; Bonchek *et al.*, 1974; Sugimura and Starr, 1977), the use of profound hypothermia seems more popular (Dillard *et al.*, 1971; Barratt-Boyes, 1973; Venugopal *et al.*, 1973; Castaneda *et al.*, 1974; Bailey *et al.*, 1976). A similar mortality and morbidity has been reported by Pooley *et al.* (1976) using standard cardiopulmonary bypass or profound hypothermia. Ischaemic cardioplegia gives the surgeon an ideal opportunity for performing accurate surgery on a motionless, relaxed heart in a bloodless field. By lowering the body temperature to 18°C, the period of tissue protection afforded even with complete ischaemia is at least 60 to 70 minutes (Barratt-Boyes, 1973). This offers the surgeon ample time to perform a totally corrective operation for many congenital defects. Comparative metabolic studies, using deep hypothermia by surface cooling and bypass cooling, have failed to establish the superiority of one or the other technique (Harris, 1973). Though deep hypothermia by surface cooling advocated by Hikasa *et al.* (1967) and Barratt-Boyes (1973) is more widely practised, our experience with perfusion induced profound hypothermia shows this to be safe even in the neonatal period.

Metabolic effects are within acceptable limits and there is a low incidence of cerebral complications. Postoperative convulsions occurred in 3 patients: 2 recovered, and 1 remains on anticonvulsant drugs. Two infants failed to regain consciousness and death was considered to be the result of air embolism caused by errors in surgical technique. Up to the end of 1973, the hypothermic technique we have described was used for all infants and children under 2 years of age who required open heart surgery. At the present time the technique is used for almost all infants under 1 year of age or 10 kg in weight and for selected cases between 1 and 2 years. Our experience suggests that profound hypothermia and circulatory arrest is particularly suitable for the correction of patients with total anomalous pulmonary venous connection and transposition of the great vessels.

A better knowledge of the pathophysiology and natural history of congenital heart diseases and the results of surgical treatment in infancy have enabled us to identify anomalies demanding early total correction. In our series, total anomalous pulmonary venous correction, simple transposition of the great vessels, symptomatic isolated ventricular septal

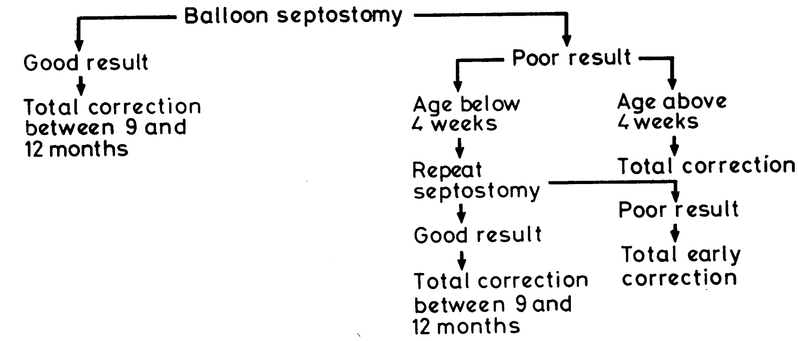

Fig. 5 *Current policy in the management of simple transposition of the great vessels.*

defect, and aortopulmonary window were the most common of these. The results of corrective surgery for total anomalous pulmonary venous connection are most encouraging and justify the policy of early correction, regardless of the age of the infant. Of 6 deaths in this group, 2 had very small left heart chambers at necropsy. Resistant pulmonary oedema was the cause of another death, though this complication has not been seen frequently in our series; the other deaths were related to technical errors and were, therefore, avoidable.

The management of infants presenting with simple transposition of the great vessels is becoming standardised throughout the world. Our policy is outlined in Fig. 5. The corrective procedure (Mustard's operation) is performed electively between the ages of 9 and 12 months. With the use of 4 to 6 ml balloon catheters for septostomy, it has seldom been necessary to consider open septectomy or correction before 9 months of age. Most of the deaths occurred early in our experience. In the last 25 consecutive cases, there has been 1 avoidable death.

The relatively small number of ventricular septal defects corrected under 1 year of age is explained by the fact that until 1974 banding of the pulmonary artery was employed in small babies in this clinic. Our current policy in the management of isolated ventricular septal defect with intractable cardiac failure and persistence or progression of pulmonary arterial hypertension is in favour of primary closure. We now reserve banding for multiple ventricular septal defects and for ventricular septal defect with associated anomalies (for example coarctation of the aorta). The presence of unsuspected multiple ventricular septal defect was the cause of 1 death. Accurate preoperative diagnosis of the anatomical site of the ventricular septal defect is of considerable importance as this may affect the surgical approach. Left ventricular cineangiography, to demonstrate the interventricular septum and any defect(s), is essential.

The successful outcome in the small group of patients with aortopulmonary window, who were in congestive cardiac failure unresponsive to medical treatment, justifies surgical intervention as soon as the diagnosis has been made. All 5 infants recovered uneventfully. One of these weighed only 2 kg at the time of operation.

Many infants with Fallot's tetralogy reach the age of 3 to 4 years without surgical intervention. Total correction is performed using conventional bypass techniques at that age. Primary early correction versus staged treatment continues to be the subject of debate. Our present policy is in favour of early total correction in the presence of cyanotic spells, if the diameter of the pulmonary arteries is adequate and the pulmonary valve ring is at least half the diameter of the ascending aorta. Where there is gross disproportion between the great vessels, we favour a pulmonary to systemic shunt, because we prefer to avoid the use of gusset reconstruction of the right ventricular outflow and pulmonary valve ring in infants with tetralogy. In our experience, pulmonary regurgitation is not well tolerated in the early postoperative period and we prefer to reconstruct severely restrictive outflow tracts after the age of 4 years by grafting a complete (tri-leaflet) aortic homograft into the right ventricular outflow tract and main pulmonary artery. The survivors after total correction in infancy are progressing well, though the follow-up time is still too short for a definitive judgement. Complete heart block, with fatal outcome, occurred in 1 patient with Fallot's tetralogy.

Complex forms of transposition of the great vessels, pulmonary atresia with intact septum, and most of the conditions listed in the miscellaneous group (Table 5) for which both early total correction and palliative operations are followed by high mortality, represent the frontier of paediatric cardiac surgery and still present a formidable challenge to cardiologists and surgeons. Successful complete repair of truncus arteriosus in a 4-month-old infant encourages us to pursue an aggressive surgical approach to this abnormality in the future.

Conclusions

Our experience in the management of critically ill infants with congenital heart disease leads us to the following conclusions.

(1) The ability to perform the complete repair of congenital heart defects in infancy is a significant advance. One method of achieving this is by the use of profound hypothermia and circulatory arrest which enables the surgeon to work on a relaxed heart in a bloodless field. This has enlarged the scope of surgical intervention, both in terms of early primary total correction and open heart palliative treatment.

(2) Earlier referral and complete diagnosis, adequate preoperative preparation, and improved surgical technique and postoperative management of these infants has been paralleled by a significant lowering of the surgical mortality.

The mortality for total correction in the neonate is double that for the infant of 6 months of age. It is our policy, therefore, to delay total correction if the clinical condition allows. In the case of total anomalous pulmonary venous connection and aortopulmonary window, however, mortality is unrelated to age and total correction is advised as soon as the diagnosis is made.

(3) The neonate and infant tolerate open heart surgery remarkably well. Whenever it has been possible to perform a complete or nearly complete anatomical repair, without the creation of complete heart block, a satisfactory result has seldom been in doubt. Complex repairs, provided they fulfil these criteria, can be equally successful.

(4) It is likely that in the future the surgical treatment of congenital heart disease will more often be performed during infancy and the first few years of life. Expertise in many disciplines and close co-operation between them is essential for success. The relatively constant annual workload in this field justifies the concentration of specialist skills in several disciplines within a limited number of properly equipped and adequately staffed units.

(5) The longer term follow-up of children who have undergone totally corrective cardiac surgery is awaited with interest. So far, results are highly encouraging as the majority of children, including those treated surgically in the neonatal and infant age range, are leading active lives and are attending normal schools. Many have no residual disabilities related to the cardiovascular system.

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